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Role of Prophylactic Anticoagulation for Direct Current Cardioversion In Patients With Atrial Fibrillation or Atrial Flutter

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The need for prophylactic anticoagulation to prevent embolism before direct current cardioversion is performed for atrial fibrillation or atrial flutter is controversial. To examine this issue further, a retrospective review was undertaken to assess the incidence of embolic complications after cardioversion. The review involved 454 elective direct current cardioversions performed for atrial fibrillation or atrial flutter over a 7 year period.

The incidence rate of embolic complications was 1.32% (six patients); the complications ranged from minor visual disturbances to a fatal cerebrovascular event. All six patients had atrial fibrillation, and none had been on anticoagulant therapy ($p = 0.026$). The duration of atrial fibrillation was <1 week in five of the six patients who had embolic complications.

Baseline characteristics of patients with a postcardioversion embolic event are compared with those of patients who did not have an embolic event. There was no difference in the prevalence of hypertension, diabetes mellitus or prior stroke between the two groups, and there was no difference in the number of patients who were postoperative or had poor left ventricular function. Left atrial size was similar between the two groups. No patient in the embolic group had valvular disease.

No patient with atrial flutter had an embolic event regardless of anticoagulant status; therefore, anticoagulation is not recommended for patients with atrial flutter undergoing cardioversion. Prophylactic anticoagulation is pivotal in patients undergoing elective direct current cardioversion for atrial fibrillation, even those with atrial fibrillation of <1 week's duration.

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Atrial fibrillation increases the risk for ischemic stroke by a factor of 5 over a lifetime. It has been estimated that one third of all patients with atrial fibrillation will eventually have a cerebrovascular accident (1). Atrial fibrillation has also been implicated in up to 37% of asymptomatic cerebral infarcts diagnosed by computed tomographic scans (2–4). Because of the incidence of stroke, some investigators (5–7) recommend that certain subgroups of patients with atrial fibrillation receive long-term anticoagulant therapy. Although the risk of embolism may be decreased by anticoagulant therapy, there is a lack of consensus regarding the need for such therapy before direct current cardioversion is performed in patients with atrial fibrillation or atrial flutter (8–10). To further assess the need for anticoagulation in the setting of elective cardioversion we retrospectively reviewed the results of 628 consecutive cardioversions.

Methods

Study patients. Adult patients undergoing elective direct current cardioversion at our institution for atrial fibrillation or atrial flutter (of at least 48 h duration) between January 1, 1983 and June 30, 1990 were included. Our analysis excluded unsuccessful cardioversions, because the conversion to normal sinus rhythm is the presumed risk factor for embolic events. We excluded 90 cardioversions on this basis, along with 51 in which atrial fibrillation or atrial flutter was present for <48 h and 33 cardioversions with indeterminate anticoagulant status. There were 628 procedures performed in the study period and 454 (in 428 patients) were subsequently reviewed. In 177 cardioversions (40.7%), the patient was taking anticoagulant medication (Coumadin in 98.3%). This report summarizes the characteristics of the patients undergoing direct current cardioversion and the embolic phenomena that occurred as a direct consequence of the procedure.

Cardioversion. All procedures were carried out in a fully equipped cardiac catheterization or electrophysiology laboratory with emergency equipment available. Digitalis preparations were withheld the morning of the procedure, and the patient was fasting. Intravenous sedation (usually a bolus injection of Brevital 0.5 mg/kg body weight) was given, and a synchronized, direct current shock was administered to restore sinus rhythm. Pre- and postcardioversion rhythm strips were available to verify the presence of atrial fibrillation or atrial flutter before the procedure and subsequent sinus rhythm after cardioversion. Cardioversions performed in the patient's room or the intensive care unit for the purpose of terminating hemodynamically unstable atrial fibrillation or atrial flutter were excluded.

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Data analysis. The medical record was reviewed for demographic characteristics and underlying diseases (hypertension, diabetes mellitus or prior cerebrovascular accident). In addition, echocardiographically documented left atrial size, as well as left ventricular function (documented by catheterization or echocardiographic analysis), was noted. The duration of the specific rhythm disturbance was analyzed. If the patient had recently undergone open heart surgery, the nature of the procedure was noted, and for valvular surgery, the position and type of valve were identified. Anticoagulant medication was recorded and laboratory variables were checked to confirm therapeutic activity. A prothrombin time >15 s (1.5 times the lower limit of the 10-s control value in our laboratory) or an activated partial thromboplastin time of 50 s (twice the lower control limit) was considered therapeutic.

Follow-up. All patients were followed up for evidence of embolism for at least 2 weeks, and postoperative patients were followed up until their 6-week postoperative visit. Embolic events were coded as having occurred in the cerebral, pulmonary or peripheral circulation and were documented by computed tomography, ventilation-perfusion scanning or angiography.

Statistics. All values are given as the mean values ± 1 SD. Group comparisons of categoric variables were made by using the chi-square or Fisher exact test, as appropriate. Continuous variables were compared by using the Student t test. A p value <0.05 was considered significant.

Baseline characteristics. The predominant rhythm disturbance requiring cardioversion was atrial fibrillation (atrial fibrillation in 72.8%, atrial flutter in 27.2%). The majority (68.6%) of the patients with atrial fibrillation were male, and the average age was 64 ± 9.7 years (range 21 to 88). Concomitant hypertension was present in 36.6% of patients, diabetes mellitus in 14.7%, a history of prior cerebrovascular accident in 10.7%. There were 206 patients (67.3%) with postoperative atrial fibrillation at the time of cardioversion. These baseline characteristics are summarized further in Figure 1. There were no statistical differences between the characteristics of patients who were or were not receiving anticoagulant therapy.

In the patients with direct current cardioversion for atrial fibrillation, the duration of arrhythmia ranged from 48 h to 7 years. The duration of atrial fibrillation was <8 days in 43.7% of cardioversions, 8 to 30 days in 29.5%, >30 days but <1 year in 25.9%, and >1 year in 0.9% of cardioversions (Fig. 2). Prosthetic valves had been implanted in 85 patients (27.7%); the prosthesis was in the mitral valve position in 44 patients, the aortic valve position in 32 and in both positions in 9.

Echocardiographic data were available for 63.3% of patients undergoing direct current cardioversion for atrial fibrillation. The average left atrial size was 49 ± 8.2 mm (range 27 to 72). The left atrial size did not correlate with the duration of arrhythmia; in patients with atrial fibrillation for <30 days the average left atrial size was 49 ± 8.2 mm and in those with atrial fibrillation for >30 days the average size was 50 ± 8 mm.

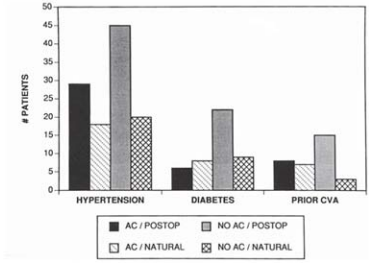


Figure 1. Baseline characteristics of the patients with atrial fibrillation. AC = with anticoagulant therapy; CVA = cerebrovascular accident; NO AC = without anticoagulant therapy; natural = patients who did not have atrial fibrillation related to an operative intervention; POSTOP = postoperative.

Assessment of left ventricular function was available for 96.1% of patients; 70.1% were characterized as having normal function or mild dysfunction. Of the remaining patients, 19.7% had moderate and 10.2% had severe left ventricular dysfunction.

Results

Atrial fibrillation. There were 454 cardioversions performed to treat atrial fibrillation or atrial flutter. Embolic events occurred in 6 of 179 cardioversions for atrial fibrillation in the absence of anticoagulant therapy. No embolic events occurred in the 153 cardioversions for atrial fibrillation in patients who were taking an anticoagulant agent. This difference was statistically significant ($p = 0.026$). Anticoagulation was used in 47% of patients without embolism and in

Figure 2. Duration of atrial fibrillation. Abbreviations as in Figure 1.

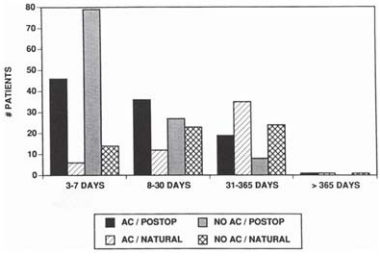


Table 1. Characteristics of Six Patients With an Embolic Event

Pt No.	Age (yr) Gender	Anticoagulant Therapy	Rhythm	Duration of AF (days)	LV Function	Postop	Event	Time to Event (days)
1	59/F	No	AF	4	Normal	Yes	Peripheral embolism	1
2	70/F	No	AF	5	Moderate	Yes	Cerebral embolism	7
3	55/M	No	AF	19	Severe	No	Pulmonary embolism	<1
4	71/M	No	AF	4	Moderate	No	Cerebellar embolism	3
5	67/M	No	AF	3	Severe	No	Cerebral embolism	4
6	77/M	No	AF	6	Mild	No	Cerebral embolism	2

AF = atrial fibrillation; F = female; LV = left ventricular; M = male; Postop = postoperative; Pt = patient.

none of the six patients with embolism ($p = 0.02$). The consequences of these embolic events ranged from minor visual disturbances to a fatal cerebrovascular embolus. In the group of six patients with embolism, four had hypertension and one patient had a history of prior cerebrovascular accident. No significant bleeding episodes related to anticoagulation were noted in the study group. The average duration of atrial fibrillation in the six patients with embolism was 6 ± 4 days (range 3 to 19) and two patients (33%) were postoperative. The average left atrial size was 44 ± 5 mm; and left ventricular function was impaired to a moderate or severe degree in four (67%) of the six (Table 1).

Cardioversion with anticoagulant therapy. There were 153 cardioversions performed on patients with laboratory confirmation of therapeutic anticoagulation. Postoperative atrial fibrillation was present in 101 cases; the remaining 52 cardioversions were performed for naturally occurring atrial fibrillation (that is, atrial fibrillation that did not occur in the postoperative period). Nineteen percent of the cardioversions were performed in patients who had been receiving anticoagulant therapy for >2 weeks before cardioversion (all except one patient were in the cohort with naturally occurring atrial fibrillation). The duration of anticoagulation did not appear to affect the incidence of embolic events.

Cardioversion without anticoagulant therapy. There were 179 cardioversions performed in patients for atrial fibrillation who were not receiving anticoagulant therapy: 115 were postoperative and 64 were in the group with naturally occurring atrial fibrillation. Two cases of embolism occurred in the postoperative group without anticoagulant therapy (incidence rate 1.7%) and four occurred in the natural atrial fibrillation group without anticoagulant therapy (incidence rate 6.2%; $p = NS$). These results are summarized in the flow diagram in Figure 3.

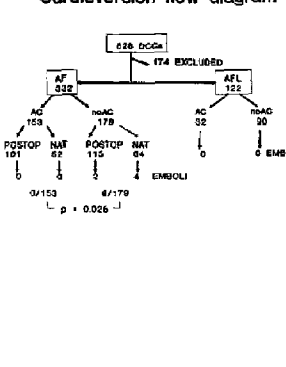
Atrial flutter. We also analyzed data from cardioversions in 122 patients with atrial flutter. Similar to others (8,11), we found no embolic events in this group, regardless of anticoagulant status (26% with and 74% without anticoagulant therapy). The number of patients with atrial flutter in our study may be too small to fully detect a low frequency event such as postcardioversion embolism. Our data support the current practice of not giving anticoagulant therapy to patients with atrial flutter who undergo cardioversion.

Discussion

Synchronized electrical cardioversion for the termination of both atrial and ventricular tachyarrhythmias in a large series of patients was first reported by Lown (12) in 1967. The most devastating complication of this procedure is systemic embolization, which has been estimated to occur in up to 7% of patients (13). The atrial arrhythmia most likely to be associated with postcardioversion embolism is atrial fibrillation (8,11), a finding further supported by our results. No patient in our series who underwent cardioversion for atrial flutter had an embolic event.

Mechanism of embolization. The mechanism of embolization in atrial fibrillation appears to be complex. Researchers (6) have documented increased cerebral blood flow after conversion from atrial fibrillation to normal sinus rhythm, which predisposes to cerebral embolism in the distribution of the middle cerebral artery. An intrinsic but unidentified property of atrial fibrillation may lead to abnormalities in clotting function, which may contribute to the incidence of embolization after cardioversion (14). Atrial fibrillation is associated with increased levels of atrial natriuretic peptide and an increase in hematocrit (15). These changes may

Figure 3. Flow diagram showing the analysis of 628 cardioversions and the patients with subsequent embolism. AC = with anticoagulant therapy; AF = atrial fibrillation; AFL = atrial flutter; DCC = direct-current cardioversion; NAT = natural atrial fibrillation (see text); noAC = without anticoagulant therapy; POSTOP = postoperative.



account for the increase in stroke with chronic atrial fibrillation.

One of the suggested mechanisms of embolism is the formation during atrial fibrillation of a clot that is expelled with the sudden resumption of mechanical atrial systole. Obarski et al. (16) detected a spontaneous echo contrast, consistent with blood stasis, during transesophageal echocardiography in a patient with atrial fibrillation, enlarged left atrium and a normal mitral valve. The study was repeated after the patient was in sinus rhythm. The echo contrast (residual stasis) was localized to the left atrial appendage, whereas the remainder of the left atrium was clear. Obarski et al. (16) theorized that this residual blood stasis may account for embolic events several days after sinus rhythm is restored. However, this residual stasis does not account for the lower incidence of embolism with paroxysms of atrial fibrillation compared with the incidence with chronic atrial fibrillation. Short paroxysms may not allow the hormonal changes that result in clot formation to occur.

Incidence of embolization. Although it has been suggested that patients with atrial fibrillation should receive anticoagulant therapy before undergoing direct current cardioversion (11,12), there is a lack of consensus on such treatment. Data are sparse on the incidence of embolism after cardioversion for specific arrhythmias. In a 1967 review, Lown (12) examined 456 cardioversion attempts and reported a 1.2% incidence rate of embolization. No patient received anticoagulant therapy at the time of the procedure. In 1969, Bjerkelund and Orning (9) reported the effect of anticoagulation on the rate of stroke after cardioversion in a nonrandomized population of 437 patients. They reported that cardioversion without anticoagulation resulted in a 5.3% incidence rate of embolism compared with a rate of 0.8% with anticoagulation ($p = 0.016$). Although the study was nonrandomized, the results are important as the patients receiving anticoagulant therapy were at higher risk for embolization because of their underlying rheumatic heart disease, congestive heart failure and history of prior embolic events. Unfortunately, because the patients were receiving long-term anticoagulant therapy, the effect of prophylactic anticoagulation before cardioversion cannot be addressed. Also, subgroups were not analyzed by rhythm (atrial fibrillation or atrial flutter), which is reported to be an independent risk factor for embolic phenomena after cardioversion (11,17,18).

More recently, Mancini and Weinberg (19) reviewed the data from the University of Michigan over a 10-year period and analyzed embolic events after cardioversion for atrial fibrillation. They found no events in the group with anticoagulant therapy, whereas 7% of the group without such therapy had embolic complications. Although the numbers are small, the results support well managed, short-term anticoagulant treatment in high risk patients (defined by the authors as patients >55 years old with coronary artery disease, cardiomyopathy or hypertension and with a duration of atrial fibrillation of >1 year).

Although our 1.32% incidence rate of embolic events after cardioversion for atrial fibrillation is similar to that of other reports (20–22), there may be a subset of patients who are at higher risk for embolic events: patients with naturally occurring atrial fibrillation who do not receive anticoagulant therapy. These patients had an incidence of embolism that was nearly four times that of the postoperative group without anticoagulant therapy. Although this difference in incidence did not achieve statistical significance, further investigation in a larger series of patients seems warranted.

Duration of anticoagulation. Atrial fibrillation of long duration is a well documented risk factor for stroke (1). It has been recommended (8,11) that patients with atrial fibrillation of short duration (≤1 week) do not need anticoagulation before undergoing cardioversion. In our patients who had an embolic event, the duration of atrial fibrillation ranged from 3 to 19 days (average 6 ± 4), with five of the six patients having atrial fibrillation for <1 week. Direct current cardioversion appeared to be hazardous in our patients with atrial fibrillation of short duration who were not receiving anticoagulant therapy. Our results suggest that low dose anticoagulation with a prothrombin time ≥15 s was protective against embolic events.

Most cases of embolism have been reported to occur within hours to a few days of cardioversion (9,10). Theoretically, however, embolism can occur several weeks after electrical conversion when mechanical atrial activity resumes (23). While this theoretical risk of embolism remains for up to 3 weeks (24,25), all of the embolic events in our patients occurred within 7 days after cardioversion. It is therefore unclear how long anticoagulant therapy should be continued after successful conversion to sinus rhythm. Because of the retrospective nature of our data, the length of time that anticoagulation was continued after cardioversion is not well documented. We cannot conclude that anticoagulation at the moment of cardioversion, without its continuation, will prevent a delayed embolism. Manning et al. (23) followed the recovery of atrial mechanical function in 21 patients over 3 months with serial Doppler echocardiographic evaluations. They found that peak *A* wave velocity and percent of atrial contribution to left ventricular filling did not return to normal until 3 weeks after cardioversion in patients who maintained sinus rhythm. Continued anticoagulation for several weeks is not likely to result in significant bleeding complications (26) and probably should be maintained until mechanical atrial systole resumes.

Our data raise a number of important issues. Should a patient with atrial fibrillation of >2 days' duration receive anticoagulant therapy before elective direct current cardioversion? Is the anticoagulant of choice Coumadin, or is heparin adequate therapy? We believe that further investigation with randomized, prospective trials should be undertaken to answer these important clinical concerns.

Limitations of study. The limitations of our study are those inherent in any retrospective review. There was no randomization of treatment arms for anticoagulation, and we

were unable to identify specific high risk subsets that would benefit from anticoagulant therapy. However, despite these limitations, all the embolic events came from the group without anticoagulant therapy.

Conclusions. In our series, atrial flutter was never associated with an embolic event and we do not recommend routine anticoagulation in these patients. Our results strongly support the use of anticoagulant treatment in patients with atrial fibrillation of at least 48-h duration who are undergoing elective synchronized direct current cardioversion. It seems prudent to continue anticoagulation until mechanical atrial systole resumes. Although others (9,13,27) have reported a similar risk of embolization with chemical cardioversion, our study was not designed to examine this risk. Further research is needed for those undergoing cardioversion with antiarrhythmic agents, as anticoagulation may be appropriate based on theoretical considerations.

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